Surgical and nonsurgical treatment of peritoneopericardial diaphragmatic hernia in dogs and cats: 58 cases (1999–2008)

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Objective—To determine clinical findings and outcomes for cats and dogs with peritoneopericardial diaphragmatic hernia (PPDH) treated surgically or nonsurgically.

Design—Retrospective case series.

Animals—28 dogs and 30 cats.

Procedures—Medical records for cats and dogs evaluated at 1 of 2 veterinary teaching hospitals were reviewed, and data regarding clinical signs, diagnostic and surgical findings, and outcome were evaluated.

Results—Prevalence of PPDH in the 2 hospitals during the study period was 0.025% (10.062% and 0.015% for cats and dogs, respectively); PPDH was an incidental finding for 13 (46.4%) dogs and 15 (50.0%) cats. Other congenital abnormalities were identified in 16 (57.1%) dogs and 7 (23.3%) cats (most commonly umbilical hernias, abdominal wall hernias cranial to the umbilicus, or sternal anomalies). Thirty-four (58.6%) animals underwent surgical repair of PPDH; 27 (79.4%) of these animals had a primary diagnosis of PPDH. Detection of clinical signs of PPDH (primary diagnosis) and intestines in the pericardial sac were significantly associated with surgical treatment. Short-term mortality rate for surgically treated animals was 8.8% (3/34). Clinical signs associated with PPDH resolved in 29 (85.3%) of surgically treated animals. No significant differences were detected between dogs and cats or between surgically and nonsurgically treated animals regarding long-term survival rate.

Conclusions and Clinical Relevance—Results of this study indicated animals with clinical signs of PPDH were more likely to undergo surgery than were animals without such signs. Herniorrhaphy was typically effective for resolution of clinical signs. Long-term survival rates were similar regardless of treatment method. Surgical or nonsurgical treatment of PPDH may be appropriate for animals with or without clinical signs, respectively. (J Am Vet Med Assoc 2013;242:643–650)

Peritoneopericardial diaphragmatic hernia is an uncommon congenital malformation that can lead to dyspnea, collapse, and death for cats and dogs.1–11 This malformation allows passage of abdominal contents through the diaphragmatic hernia into the pericardial sac. Although the developmental abnormality responsible for the malformation has not been identified, PPDH is likely caused by failure of formation or fusion of the septum transversum.2,3 Proposed mechanisms for development of this hernia include prenatal trauma to the diaphragm.2,4,7,15–18 Other authors4–6 have reported that mature adult and geriatric cats have a higher rate of incidental diagnosis of PPDH versus young cats; however, few reports of the clinical signs and prognosis for animals with PPDH have been published. Although there are many reports4–6,8–40 of animals with PPDH, little information has been published regarding optimal methods of treatment for such animals. Treatment rec-
oomendations for animals with PPDH are often made on the basis of clinical signs; to the authors’ knowledge, no studies have been conducted to evaluate this method of treatment selection.

The objective of the study reported here was to determine the clinical signs, physical examination and radiographic findings, and short- and long-term outcomes after surgical or nonsurgical treatment of cats and dogs with PPDH. On the basis of our clinical impression, we hypothesized that a higher percentage of cats and dogs with clinical signs of PPDH would be treated surgically versus cats and dogs without clinical signs. Additionally, we hypothesized that the long-term survival rates for cats and dogs with PPDH treated surgically would be similar to those for cats and dogs with PPDH treated nonsurgically.

Materials and Methods

Case selection—Medical records of dogs and cats with PPDH evaluated at 1 of 2 veterinary teaching hospitals (The Ohio State University and University of Wisconsin) from June 1999 through June 2008 were identified via search of the medical records database of each hospital. Data determined from the medical records included species, breed, age, sex, body weight at the time of diagnosis, clinical signs, physical examination findings, results of blood analyses, results of additional diagnostic tests (including thoracic and abdominal radiography, ultrasonography, and CT), and information regarding surgical procedures, medical recommendations, short-term (<2 weeks after the date of surgery) and long-term (≥2 weeks after the date of surgery) surgical complications, and cause of death. The PPDH diagnosis for each animal was determined to be primary or incidental on the basis of medical history, clinical signs, and physical examination findings. A diagnosis of PPDH was determined to be incidental for animals with minimal to no clinical signs and if a diagnosis was made during diagnostic evaluation for clinical signs primarily attributable to another disease or condition. A diagnosis of PPDH was determined to be a primary diagnosis for animals with clinical signs attributable to PPDH (ie, exercise intolerance, tachypnea, dyspnea, cough, or vomiting). Prevalence of PPDH was determined by dividing the number of cats or dogs with PPDH by the total number of cats or dogs evaluated at the 2 hospitals during the study period. Follow-up information was obtained via telephone interviews of owners or referring veterinarians.

Inclusion criteria—Cats and dogs for which complete medical records were available and a diagnosis of PPDH had been made on the basis of results of radiography, ultrasonography, CT, or exploratory surgery were included in the study. Cats and dogs were excluded from the study if results of radiography or ultrasonography had been suggestive of PPDH but results of subsequent exploratory surgery had not confirmed that diagnosis.

Radiographic assessment—Lateral and ventrodorsal or dorsoventral thoracic radiographic views were evaluated by 2 investigators (CGB and MSB). Information regarding skeletal abnormalities (abnormal number of sternebrae, pectus excavatum, kyphosis, or lordosis), cardiac size, abdominal tissues identified in the pericardial sac (eg, intestines, liver, or spleen), detection of a mesothelial remnant, appearance of the lungs, and ability or inability to distinguish between the diaphragm and cardiac silhouette were determined via evaluation of radiographic images.

Statistical analysis—Descriptive data were reported as range, median, and mean ± SD values. Data were analyzed via the Student t test and χ² test. Values of P < 0.05 were considered significant.

Results

Prevalence of PPDH and demographic data—Twenty-eight dogs and 30 cats were included in the study. The prevalence of PPDH for cats (30/48,685 [0.062%]) was significantly (P = 0.04) greater than it was for dogs (28/185,822 [0.015%]) in the 2 veterinary teaching hospitals during the study period; the overall prevalence of PPDH in cats and dogs was 0.025%. The most common breeds of dogs and cats with PPDH were Weimaraner (6/28 [21.4%]) and domestic longhair (10/30 [33.3%]), respectively. Median age at the time of PPDH diagnosis was not significantly different between dogs (1.2 years; range, 0.12 to 12.30 years) and cats (1.0 year; range, 0.25 to 12.30 years). No significant sex predisposition for PPDH was identified; however, more males than females had PPDH (16/28 [57.1%] dogs and 19/30 [63.3%] cats were male). Forty animals (69.0%; 22/30 [73.3%] cats and 18/28 [64.3%] dogs) had clinical signs of PPDH. Historical clinical signs that could be attributed to PPDH were the primary reason for initial examination for 15 (50.0%) cats and 15 (53.6%) dogs in the study; these animals were determined to have a primary diagnosis of PPDH. Therefore, 13 (46.4%) dogs and 15 (50.0%) cats had an incidental diagnosis of PPDH. The most common clinical signs in animals with a primary diagnosis of PPDH included exercise intolerance, tachypnea, dyspnea, cough, vomiting, and anorexia. Tachypnea or dyspnea was the most common clinical sign for dogs and cats with PPDH (Table 1).

Table 1—Clinical signs in dogs and cats with PPDH that were treated surgically or nonsurgically.

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>Dogs (n = 28)</th>
<th>Cats (n = 30)</th>
<th>Cats and dogs that underwent surgery (n = 34)</th>
<th>Cats and dogs that did not undergo surgery (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tachypnea or dyspnea</td>
<td>10 (35.7)</td>
<td>20 (66.7)</td>
<td>16 (47.1)</td>
<td>7 (29.2)</td>
</tr>
<tr>
<td>Exercise intolerance</td>
<td>6 (21.4)</td>
<td>6 (20.0)</td>
<td>9 (26.5)</td>
<td>3 (12.5)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>2 (25)</td>
<td>8 (26.7)</td>
<td>11 (32.4)</td>
<td>4 (20.0)</td>
</tr>
<tr>
<td>Anorexia</td>
<td>5 (17.9)</td>
<td>4 (13.3)</td>
<td>5 (14.7)</td>
<td>5 (20.8)</td>
</tr>
</tbody>
</table>

Data are number (%).
Peritoneopericardial diaphragmatic hernia was considered an incidental diagnosis for animals evaluated because of reasons related to other disease processes such as vehicular trauma, neurologic disease, orthopedic disease, pancytopenia, chylothorax, hyperthyroidism, and mastocytosis; a diagnosis of PPDH was made for these animals on the basis of evaluation of thoracic radiographic images or images obtained via other imaging modalities.

Results of physical examination—The most common physical examination findings for animals with PPDH were heart murmur (6 dogs and 8 cats), muffled heart sounds (10 dogs and 4 cats), and decreased lung sounds (5 dogs and 5 cats). Seven cats and 2 dogs had increased respiratory effort. Borborygmus was auscultated in the thoracic cavity of 4 dogs and none of the cats. Three cats had neurologic signs (1 each nystagmus, signs of lumbosacral disease, and signs of cauda equina syndrome), and 2 dogs had tetraparesis secondary to caudal cervical spondylomyelopathy.

Sixteen of the 28 (57.1%) dogs and 7 of the 30 (23.3%) cats with PPDH had evidence of other congenital abnormalities that were detected during physical examinations performed before or at the time of PPDH diagnosis. The most common of these abnormalities were umbilical hernia or supraumbilical hernia (ie, abdominal wall hernia cranial to the umbilicus; n = 12 dogs and 3 cats), cryptorchidism (2 dogs and 1 cat), cleft palate (1 dog), and persistent pupillary membranes (2 dogs).

Results of diagnostic testing and imaging—Congenital abnormalities other than PPDH detected via diagnostic imaging included intrahepatic PSS (2 dogs), extrahepatic PSS (1 cat), other cardiovascular defects (5 dogs), and sternal or vertebral anomalies (11 dogs and 3 cats); Table 2. A CBC and serum biochemical analyses were performed for 25 cats and 19 dogs with PPDH. The most common hematologic abnormality for dogs was a serum alanine aminotransferase activity higher than the reference range (n = 10), and the most common hematologic abnormality for cats was a serum calcium concentration higher than the reference range (9). Ventrodorsal and lateral thoracic radiographic images were obtained for 55 animals (29 cats and 26 dogs). The most common radiographic abnormalities were an enlarged cardiac silhouette and a loss of the distinction between the heart and the diaphragm attributable to superimposed soft tissue or fat opacities (Figure 1). Additional diagnostic imaging was performed for 17 dogs and 22 cats. Abdominal ultrasonography was performed for 7 dogs and 4 cats, echocardiography was performed for 13 dogs and 18 cats, thoracic ultrasonography was performed for 4 dogs and 1 cat, and CT was performed for 1 dog. Echocardiography reports for 31 of the animals (13 dogs and 18 cats) indicated that a complete evaluation of the heart could not be completed because of an abnormal heart position or compression of the heart by herniated abdominal organs and tissues. Primary cardiac defects were identified in 9 cats and included hypertrophic cardiomyopathy (n = 3), double-chambered right ventricle with pulmonic stenosis (1), mitral stenosis with left atrial thrombus (1), tricuspid regurgitation (1), mitral regurgitation (1), left ventricular wall thickening (1), and dynamic right ventricular outflow obstruction (1). Primary cardiac defects were identified in 4 dogs and included ventricular septal defects (n = 3), mitral regurgitation (1), persistent left cranial vena cava (1), tricuspid dysplasia (1), and subaortic stenosis (1).

Surgical treatment of PPDH—Thirty-four (58.6%) animals (19 dogs and 15 cats) underwent surgical repair

<table>
<thead>
<tr>
<th>Variable</th>
<th>Dogs*</th>
<th>Cats*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Surgery (n = 18)</td>
<td>No surgery (n = 8)</td>
</tr>
<tr>
<td>Enlarged cardiac silhouette</td>
<td>18 (100)</td>
<td>7 (87.5)</td>
</tr>
<tr>
<td>Increased soft tissue or fat opacity between heart and diaphragm</td>
<td>18 (100)</td>
<td>4 (50)</td>
</tr>
<tr>
<td>Intestinal loops in pericardial sac</td>
<td>10 (55.6)</td>
<td>2 (25)</td>
</tr>
<tr>
<td>Mesothelial remnant</td>
<td>1 (5.6)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Sternal anomalies</td>
<td>3 (18.3)</td>
<td>2 (25)</td>
</tr>
<tr>
<td>Vertebral anomalies</td>
<td>1 (5.6)</td>
<td>1 (12.5)</td>
</tr>
</tbody>
</table>

Data are number (%). Findings were determined on the basis of evaluation of lateral and ventrodorsal or dorsoventral radiographic images.

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of PPDH. The median age at the time of PPDH diagnosis for animals that underwent surgery was 1.1 years (range, 0.12 to 12.30 years); age was not significantly different (P = 0.11) between cats and dogs that underwent surgery. Twenty-seven of the 34 (79.4%; 14 dogs and 13 cats) animals that underwent surgical repair of PPDH had clinical signs attributable to PPDH (primary diagnosis); 7 (20.6%) animals that underwent surgery did not have such clinical signs. For 3 of these animals, PPDH was identified incidentally and surgically repaired during celiotomy for splenectomy (n = 1 dog), ovariohysterectomy (1 dog), or umbilical hernia repair (1 dog). Loops of intestine were incidentally identified in the pericardial sac of 3 animals (2 dogs and 1 cat) that did not have clinical signs related to the problem; these animals subsequently underwent surgical repair of PPDH. One cat without clinical signs of PPDH underwent exploratory celiotomy and intestinal biopsy at the request of the owner because of anorexia and weight loss; during surgery, a PPDH was identified and portions of the liver were found in the pericardial sac.

A ventral midline celiotomy was performed for all 34 animals in this study that underwent surgical repair of PPDH (Figure 2). The tissues most frequently herniated through the PPDH included omentum (9 dogs and 4 cats), liver lobes (9 dogs and 13 cats), gallbladder (4 dogs and 10 cats), and portions of small intestine (8 dogs and 10 cats). Adhesions of the intestine or liver to the pericardium were found in 7 cats. A partial caudal sternotomy was performed for 5 of these cats so that the adhesions could be debrided. Adhesions were not detected in dogs. A partial caudal sternotomy was performed for improved surgical access in 2 dogs. One of these dogs had PPDH and chylothorax; PPDH herniorrhaphy and thoracic duct ligation and cysterna chilis ablation were concurrently performed for this dog. One of these dogs underwent reconstruction of the diaphragm with pericardium because of agenesis of approximately 60% of the diaphragm.

Figure 2—Intraoperative photographs of a ventral midline celiotomy approach (A) and herniorrhaphy (B) of a representative cat with PPDH.
The PPDH herniorrhaphies were performed with absorbable or nonabsorbable suture material, in interrupted or continuous suture patterns (Figure 2). The number of animals for which debridement of hernia margins was performed prior to PPDH repair could not be determined via evaluation of surgery reports. A thoracostomy tube was placed for 16 animals (9 cats and 7 dogs); median time that thoracostomy tubes were in place in these animals after surgery was 22.5 hours (range, 1 to 92 hours). Median hospitalization time of animals after herniorrhaphy was 3 days (range, 2 to 9 days). Identification of intestinal loops in the pericardial sac via diagnostic imaging (21/34 animals that underwent herniorrhaphy; \( P < 0.001 \)) and a primary diagnosis of PPDH (27/34 animals that underwent herniorrhaphy; \( P < 0.001 \)) were significantly and positively associated with performance of herniorrhaphy for animals.

Intraoperative complications were detected in 4 cats. Severe hemorrhage occurred in 3 cats that had adhesions of the liver to the pericardium. Two of these cats received a blood transfusion (1 of which underwent partial hepatectomy); both of these cats survived to discharge from the hospital. One cat (age, 4 months) with intraoperative hemorrhage died within 3 hours after surgery; this cat did not receive a blood transfusion. One cat developed intraoperative hypotension unassociated with blood loss, which was successfully treated. No intraoperative complications were detected in dogs.

Postoperative complications were detected in 3 dogs and 4 cats. These complications included respiratory arrest (1 cat), regurgitation (2 dogs), pericardial stenosis (1 dog), chylothorax (1 cat), incisional dehiscence (1 cat), and tracheitis (1 cat). One of the dogs with regurgitation developed esophageal stricture and subsequently underwent balloon dilation of the esophagus. The other dog with regurgitation developed hypertension, poor oxygenation (oxygen saturation as measured by pulse oximetry \( \leq 90\% \)), pleural effusion, and a tension pneumothorax; that dog was euthanized. Findings of postmortem examination of this dog indicated intrahepatic PSS. Surgical repair of the diaphragm resolved clinical signs associated with PPDH in 29 of 34 (85.3%) animals. Five (14.7%) animals that underwent surgical repair of PPDH had clinical signs consistent with PPDH after surgery. These clinical signs included development of severe esophagitis resulting in esophageal strictures (1 dog), wheezing (1 cat; this cat had a history of asthma), dyspnea secondary to chylothorax (1 cat), and intermittent vomiting (2 cats and 1 dog; the frequency of vomiting seemed to be similar before and after surgery for these 3 animals).

Nonsurgical treatment of PPDH—Twenty-four (41.4%) animals (9 dogs and 15 cats) did not undergo surgical repair of PPDH. Median age at the time of PPDH diagnosis for these animals was 7.3 years (range, 0.58 to 15.0 years); age at the time of diagnosis was not significantly \( (P = 0.82) \) different between cats and dogs that did not undergo surgery. The median age of animals that did not undergo surgical repair of PPDH was significantly \( (P < 0.001) \) greater than the median age of animals that underwent surgical repair of PPDH. Animals that did not undergo surgical repair of PPDH had significantly \( (P = 0.04) \) fewer clinical signs attributable to the problem at the time of the initial evaluation, compared with animals that underwent herniorrhaphy. Of the 24 animals that did not undergo surgical repair of PPDH, 11 (45.8%) did not have clinical signs attributable to that problem at the time of the initial evaluation and 4 (16.7%) had \( \geq 3 \) clinical signs at that time.

Two dogs and 2 cats had clinical signs attributable to PPDH but did not undergo herniorrhaphy. Reasons that these animals did not undergo herniorrhaphy included severe renal disease (1 cat) and euthanasia because of pancytopenia (1 dog); owners declined surgery for 1 cat and 1 dog. Treatments were not prescribed for treatment of PPDH for any of these animals; clinicians recommended that owners monitor the animals for signs of respiratory distress or gastrointestinal tract disorders. Medications were prescribed for these animals for treatment of other concurrent diseases. One 15-year-old cat that initially had no clinical signs of PPDH developed lethargy and dyspnea within 2 months after the diagnosis was made. Diagnostic imaging was repeated for this cat during follow-up examinations; results indicated increased compression of the heart by a structure with fat opacity and effusion in the pericardial sac. Surgery was recommended for this cat but had not been performed by the end of the study period.

Follow-up information—Mean \( \pm SD \) follow-up time after diagnosis of PPDH was significantly \( (P = 0.04) \) longer for cats (868.3 \( \pm \) 869.3 days) than it was for dogs (282.9 \( \pm \) 491.2 days). No significant \( (P = 0.06) \) differences were detected for follow-up time between animals that underwent surgery (mean \( \pm SD \), 329.5 \( \pm \) 596.1 days; range, 2 to 2,657 days) versus those that did not undergo surgery (mean \( \pm SD \), 721.2 \( \pm \) 857.3 days; range, 0 to 2,465 days). A significant \( (P < 0.001) \) difference was detected for age at the time the last follow-up information was obtained between animals that did not undergo surgery (median age, 8.56 years; age range, 0.70 to 19.74 years) and animals that underwent surgery (median age, 2.29 years; age range, 0.17 to 12.31 years). However, no significant differences were detected for age at the time the last follow-up information was obtained between cats and dogs that underwent surgical repair of PPDH (median age, 3.73 and 2.21 years, respectively; \( P = 0.15 \)) and between cats and dogs that did not undergo surgical repair of PPDH (median age, 8.98 and 8.49 years, respectively; \( P = 0.41 \)). No significant differences were detected regarding long-term survival rates between dogs and cats (\( P = 0.81 \)) or between animals that underwent surgery and those that did not undergo surgery \( (P = 0.13) \). Of the 58 animals in this study, 30 (51.7%; 17 cats and 13 dogs) were alive at the end of the study period.

Information regarding time and cause of death was available for 22 animals. Six animals (3 cats and 3 dogs) that underwent surgical repair of PPDH died. Three of these animals died within 2 weeks after surgery; therefore, the short-term mortality rate for animals that underwent herniorrhaphy was 8.8% (3/34). Causes of death for these animals included postoperative hemorrhage \( (n = 1) \), acute respiratory arrest \( (1) \), and complications attributable to an undiagnosed intrahepatic PSS \( (1) \). Three of the animals that underwent herniorrhaphy died during the long-term follow-up period; causes...
of death for these animals included pericardial stenosis and jejunal necrosis (n = 1), chylothorax (1), and a single congenital PSS (1).

Sixteen of the 24 animals that did not undergo surgical repair of PPDH died. Causes of death for these animals included euthanasia because of reasons unrelated to PPDH (4 cats and 6 dogs) and sudden death or owner-reported natural causes of death (4 cats and 2 dogs).

The 2 animals (1 dog and 1 cat) with clinical signs of PPDH that did not undergo herniorrhaphy (because owners declined surgical treatment) were alive and had no clinical signs of PPDH at the end of the study period (follow-up time, 892 days and 1,034 days for the dog and the cat, respectively). Six animals (2 cats and 4 dogs) were lost to follow-up.

Discussion

Peritoneopericardial diaphragmatic hernia is the most common congenital anomaly of the pericardium and diaphragm in dogs and cats. Animals with this anomaly can have various clinical signs that are attributable to the types and amounts of abdominal organs and tissues that herniate into the thorax; clinical signs of PPDH may be absent or the condition may be severe enough to cause death as a result of cardiac, respiratory, or gastrointestinal tract compromise. Clinical signs of animals in the present study were similar to those of animals with PPDH in other reports. A higher percentage of animals with clinical signs of PPDH underwent surgery versus animals without clinical signs in the present study. Long-term survival rate was not significantly different between animals that were treated surgically versus those that were treated nonsurgically. Multiple factors may have been considered by veterinarians regarding treatment of animals in this study, but presence or absence of clinical signs attributable to PPDH seemed to be an appropriate variable with which to make treatment recommendations for cats and dogs.

The prevalence of PPDH was higher for cats than it was for dogs, and Weimaraners and domestic long-haired cats seemed to be overrepresented in this study; these results were similar to those of other studies. Results of another study indicate the prevalence of PPDH may be higher in Persian cats than it is in other breeds of cat. Congenital agenesis of all or part of the diaphragm has an autosomal-recessive mode of inheritance in cats. The authors’ knowledge, it is not known whether PPDH is genetically inherited or whether it develops because of in utero conditions. Results of the present study indicated the prevalence of PPDH in dogs and cats was low, similar to results of other studies.

In this study, young cats and dogs (median age, 1.1 years) were more likely to have a primary diagnosis of PPDH and undergo surgical correction of the hernia compared to old cats and dogs (median age, 7.3 years) that had minimal or no clinical signs related to PPDH. To the authors’ knowledge, similar findings have not been previously reported. This relationship between age and clinical signs of PPDH may be related to sizes of hernias or amounts or types of herniated organs in young versus old animals. Large defects in the diaphragm allow organs to pass freely into the pericardial sac, which may result in adhesions of abdominal organs to the pericardium and entrapment of organs in the pericardium, leading to respiratory, cardiac, or gastrointestinal tract problems. Small defects in the diaphragm may be occluded with falciform ligament fat or omentum; animals with such defects may never have clinical signs of PPDH. This theory was supported by the finding of the present study that animals that did not undergo herniorrhaphy were older than animals that underwent herniorrhaphy and typically only fat opacities were identified in the pericardial sac of such animals (ie, abdominal tissues other than fat were not identified). One cat that did not have clinical signs of PPDH at the time of the initial examination subsequently developed respiratory tract compromise attributable to an increase in the amount of herniated tissue in the pericardial sac. Therefore, animals that do not initially have clinical signs of PPDH should be monitored for development of such clinical signs because small hernias of the diaphragm may entrap abdominal organs and tissues.

The mortality rate of animals after surgical repair of PPDH repair is reportedly high, primarily because of reexpansion pulmonary edema and hemorrhage. The short-term mortality rate (8.8%) of animals with PPDH that underwent herniorrhaphy in the present study was lower than the mortality rate (14%) of cats with PPDH that underwent herniorrhaphy in another study. However, the mortality rate (5.1%) of cats and dogs with PPDH that underwent herniorrhaphy in another study was similar to that determined in the present study. The low mortality rates for animals that underwent surgical repair of PPDH in the present study and that other recent study may be attributable to improved anesthetic and postoperative management. In particular, clinicians attempted to allow slow and controlled reexpansion of the lungs in many animals in the present study to minimize development of pulmonary edema. A thoracostomy tube was placed in 9 cats and 7 dogs in this study. Most of those cats with a thoracostomy tube underwent median sternotomy to facilitate dissection of adhesions in the pericardial sac or to facilitate replacement of abdominal organs into the peritoneal cavity. Although adhesions were not detected in dogs, a median sternotomy was performed for some dogs to facilitate replacement of abdominal organs into the peritoneal cavity. Some of the dogs with a thoracostomy tube underwent median sternotomy because repair of their diaphragmatic hernias required use of advanced reconstruction techniques. Use of thoracostomy tubes did not seem to affect outcome of animals. Thoracostomy tubes may be useful for monitoring of amount of fluid or hemorrhage in the pleural sac or for intrapleural administration of analgesic drugs. Complications of thoracostomy tubes can develop, and use of such tubes can prolong the period of hospitalization and increase morbidity of animals and costs to clients. Evaluation of all patient factors and surgical findings is recommended prior to placement of a thoracostomy tube after surgical repair of PPDH. Standard herniorrhaphy methods for repair of diaphragmatic defects do not re-
quire placement of a thoracostomy tube. However, if
the pleural sac is entered during sternotomy, if dissec-
tion of adhesions from the pericardial sac results in dis-
ruption of the pericardium, or if repair of large defects
in the diaphragm requires use of advanced reconstruc-
tion techniques, placement of a thoracostomy tube is
recommended. Furthermore, placement of thoracos-
tomy tubes may be required for treatment of animals
with concurrent pleural space disease (ie, chylothorax).

Although PPDH does not consistently cause clini-
sical signs in all animals with the problem, results of this
study were similar to those of other studies that
animals with PPDH frequently have other congenital
abnormalities that may be detected during physical
examination. For animals with supraumbilical hernia,
umbilical hernia, or cryptorchidism, owners should be
questioned regarding clinical signs of respiratory or gas-
trointestinal tract distress of the patient to help deter-
mine whether additional diagnostic testing for PPDH is
indicated. Although some animals in this study under-
went imaging techniques other than radiography, the
most commonly used diagnostic imaging method for
detection of PPDH was thoracic radiography. A diag-
nosis of PPDH was made via radiography for most ani-
mals, regardless of whether other imaging techniques
had been performed. Imaging techniques other than
radiography may be useful for identification of concur-
cent cardiac anomalies and for identification of vascular
and other soft tissue structures in the pericardial sac.

Three animals in this study had severe clinical signs
attributable to PSS. Concurrent PPDH and PSS has been reported for 2 other animals, suggesting a
possible relationship between these diseases. Clinical
signs of PPDH may be similar to those of PSS. On the
basis of results of the present study regarding morbidity
of animals with PSS that had initially been treated only
for PPDH, performance of preoperative abdominal
ultrasonography and bile acids testing or careful and
complete surgical exploration of the abdomen during
herniorrhaphy is recommended.

Most (14/19 dogs and 13/15 cats) animals in the
present study that underwent herniorrhaphy had clinical
signs attributable to a primary diagnosis of PPDH. Surgi-
cal repair of PPDH resolved clinical signs of tachypnea,
dyspnea, exercise intolerance, vomiting, and anorexia in
29 (85.3%) of these animals. Respiratory or gastrointesti-
nal tract signs were detected after surgery for 4 cats and 1
dog. Because this study was conducted retrospectively, it
was not possible to definitively determine whether these
clinical signs were attributable to PPDH, the surgical re-
pair, or to concurrent or new disease processes. A long
duration of intestinal herniation, distention, or obstruc-
tion may have caused intestinal motility disorders lead-
ing to vomiting or regurgitation before or after surgery.
Additionally, other disease processes (eg, inflammatory
bowel disease, megaeosophagus, heart disease, pleural
space disease [chylothorax or neoplasia], pulmonary
disease [bronchitis or asthma], renal disease, or liver
disease) could have caused pre- or postoperative clinical
signs. Thus, some animals in this study could have been
miscalculated as having clinical signs attributable to a
primary diagnosis of PPDH. Recurrence of PPDH was
not detected for any of the animals in this study. Echocar-
diography or thoracic radiography had been performed
after surgery for 9 (26.5%) of the animals that underwent
surgical repair of a PPDH; results of those tests indicated
hermias had been successfully repaired without recur-
rence. Thus, despite surgical and anesthetic risks associ-
ated with PPDH repair, herniorrhaphy seemed to be an
effective treatment for resolution of clinical signs in cats
and dogs with PPDH.

This study had several limitations, including the
retrospective design and the fact that follow-up times
differed among animals. Treatments were not adminis-
tered in a blinded manner, and control animals were
not included in the study. Some animals for which a
diagnosis of PPDH was made may not have had that
condition; such misclassification of animals may have
been caused by limitations of the diagnostic methods
used. However, a diagnosis of PPDH was confirmed for
all animals that underwent surgery in this study. Ad-
ditionally, respiratory or gastrointestinal tract signs in
some animals could have been attributable to problems
other than PPDH.

Results of this study supported our hypothesis that
animals with clinical signs attributable to PPDH were
more likely to undergo surgical treatment than animals
with PPDH that did not have clinical signs. Results sug-
gested that animals with PPDH that do not have clinical
signs at the time of diagnosis can develop clinical signs
at a later time. No significant differences were detected
between animals that underwent herniorrhaphy for
treatment of PPDH and those that did not undergo sur-
gery regarding long-term survival rates. The mortality
rate for animals that underwent surgery was low, and
clinical signs attributable to PPDH resolved in most of
those animals. Therefore, it may be appropriate to make
recommendations regarding surgical treatment of cats
and dogs with PPDH on the basis of the presence or
absence of clinical signs.

a. Office Excel, Microsoft Corp, Redmond, Wash.

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